Letter to the Editor

CEREBROVASCULAR DISEASE AND EXECUTIVE DYSFUNCTION IN GERIATRIC DEPRESSION

To the Editor:

In their article recently published in the Journal, Mast and colleagues (1) suggest that executive dysfunctions modulated the relationship between cerebrovascular risk factors and depressive symptoms in a population of 77 elderly patients admitted to a geriatric rehabilitation hospital.

We would like to contribute to this topic with our own personal data referring to a similar population of 209 elderly patients admitted to a Rehabilitation and Aged Care Unit from March 20001 to March 2004. Patients were selected among those who consecutively underwent a neuropsychological examination because they were suspected of having a cognitive impairment, were aged 65 years or older, and had a computed tomography (CT) of the brain. Depressive symptoms were assessed with the 15-item Geriatric Depression Scale (2). The presence and severity of cortical, white matter, and deep subcortical lesions and of leukoaraiosis were assessed on CT film with a standardized visual rating scale, which has been previously validated and used in elderly patients (3,4). With this method, the patients were quantitatively divided into two groups (50th percentile) according to the severity of subcortical cerebrovascular disease (sCVD): One hundred four patients had none or mild, and 105 had moderate or severe sCVD.

Table 1 shows that patients in the group with higher sCVD were older, more depressed, and more deteriorated both in global cognitive (as evaluated with the Mini-Mental State Examination) and in executive functions (lower performances on semantic verbal fluency and higher percentage of perseverative errors). Furthermore, they had significantly lower performances in comparison to the other group on the test measuring sustained attention.

Our data support the hypothesis that sCVD is etiologically involved in the relationship between depressive symptoms and executive dysfunctions in elderly persons. This is particularly relevant to the model proposed by Mast and colleagues because they did not use neuroimaging data, as acknowledged in the limitations of their study. Indeed, neuroimaging is necessary to confirm the vascular etiology, because presence of cardiovascular risk factors does not automatically mean presence of cerebrovascular disease. Although T2-weighted magnetic resonance imaging is the most sensitive instrument to detect sCVD in the brain, the use of a CT-based rating scale has been repeatedly shown to be reliable and specific (3,4); furthermore the scale offers the advantage to be low cost and easy to do even in low-technology settings (such as rehabilitation units). We suggest that a systematic rating of the subcortical vascular lesions on CT brain will become a routine procedure in the study of the relationship between sCVD, depression, and executive dysfunctions.

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